THE BRITISH VETERINARY JOURNAL

Vol. 109 **April, 1953** **No. 4**

QUEEN MARY

Queen Mary died peacefully in her sleep late in the evening of March 24, 1953. Thus was the passing of a great and gracious lady, whose loss will be felt with sincere grief and sorrow throughout the British Empire. By her life and work and above all by her kindness and thought for others she had earned a place in our hearts such as is normally reserved for a dearly loved mother. She was indeed the mother of all the British peoples, whatever their race or creed. She and her husband, the beloved King George V, did more to cement the Commonwealth into one large and intimate family than did any others.

She was regal in the truest sense of the word and carried her high rank and position with great dignity and poise yet she had the common touch and could enter the home of her humblest subject as a welcome guest. In her own home and family life she set an example to all. Her life had its full measure of grief and sorrow yet her sense of the duty required by her high office was so great that personal feelings always gave place to those of service to the state. Time and men come and go and memories fade but the record of the life and work of Queen Mary will be registered in history in indelible letters of gold.

EDITORIAL

NATURE MUST BE WOOED, AND NOT ASSAILED

THE ordinary succession of changes in Nature are so well ordered that they escape the notice of all, except the few who have "eyes that can see and appreciate." The marvellous precision of these changes excels anything that our scientists have discovered or evolved.

Those who have been privileged to witness the last half-century, or have knowledge of the scientific achievement of that period, are apt to forget the above fact and imagine that man's puny ingenuity is paramount. On the surface, and only on the surface, it would appear that all progress is dependent upon those who are ranked as scientists. To some extent this is true in regard to those men who have been content to spend their time unfolding the secrets of Nature and in contriving the various mechanical and other devices which have made life easier for their fellow men. Scientists have in many respects become the

(The March issue of The British Veterinary Journal was published on March 18)

masters of our life and time, and their influence has extended into spheres which in the past were considered to be beyond their province, and they have arrogated unto themselves an authority which extends even into the realms of politics and religion.

Science has done much to disclose the mysteries of life in the animal and vegetable kingdoms, and as a result we are in possession of vital information relating to the physiology of the body from conception to death. In recent years the study of the endocrine system, and in particular its influence on growth and function, has received great attention. In the normal course of events, and when nutrition and environment are propitious, the intricate mechanism of the animal body works as a fully balanced, co-ordinated whole, stimulated and regulated by products elaborated by the endocrine glands. The various sections of the endocrine system are dependent one upon the other, and the slightest aberration in one part may have widespread effects and bring chaos.

The work of the endocrinologist and biochemist has progressed to such a stage that we can give some explanation for many syndromes which are concomitant upon an imbalance in the endocrine system, and especially of those associated with certain hypo- and hyper-functional activities.

We know that in some instances a degree of resolution can be obtained by medical or surgical means and the patient can be restored to a state of apparent normality. There are instances when certain dysfunctions can be relieved by attention to the diet, for the endocrine glands, like all other tissues of the body, function best when nutritional standards are at optimum level. The intestinal flora also has its part to play in the maintenance of good health. The numbers and variety of organisms in the intestinal tract are legion, and they play a vital part in the digestive and eliminative processes. Some are pathogenic, others may under certain circumstances assume a harmful role, but normally the ecological balance is so good that the good and bad live together in reasonable amity and expend their energies on the intestinal content alone.

A continuous battle is raged, and it would appear that if one could inhibit the deleterious action of those capable of doing harm, then the process of digestion and absorption would be enhanced and to the benefit of the growth and maturity of the host.

Experiments have shown that some of the antibiotics can act in this way. Now, this is a direct interference with a natural process, and clinicians have ample evidence that when man interferes with the course of Nature, evil in some form or other may be the result. The troubles of the modern dairy cow are a striking example of outraged Nature. To correct an endocrine dysfunction in an individual animal is sound practice, but to disturb the tempo of metabolism by artificial means on whole groups of animals may bring its troubles in repercussion. When the change is brought about by the action of potent antibiotics on the intestinal flora one risk comes immediately to mind. Some of the organisms may react to the new environment, acquire a resistance to the antibiotic, and even change in their way of life and action. This would upset the ecological balance in the intestinal tract and bring trouble in its trail.

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GENERAL ARTICLES

STUDIES ON DISINFECTION OF EGGS AND INCUBATORS

I.—The Survival of *Salmonella pullorum, thompson* and *typhi-murium* on the Surface of the Hen's Egg and on Incubator Debris

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Introduction

A SEARCH of the literature has revealed no data on the length of survival of Salmonellæ on contaminated whole eggs placed in forced draft incubators. Gwatkin (1926) records the survival of *S. pullorum* for 12 days on pieces of sterilised shell stripped of the membrane and kept in an incubator at 37° C. Graham (1941) contaminated egg shells with *S. pullorum* and the organism was alive after 180 minutes' exposure in a forced draft incubator. *S. pullorum* died rapidly on pieces of sterilised shell when placed in an incubator and Gwatkin (1947) concluded that such pieces of infected shell were not satisfactory for long exposure experiments. Thus 12 of 27 pieces of infected shell placed in an incubator, yielded a growth when planted into broth, while all of similar shells kept at room temperature gave growth.

Gregory (1948) using a bacteriological incubator at a temperature of 100° F. and a wet bulb reading of 82°-86° F., found that two of 26 turkey eggs contaminated with *S. typhi-murium* remained infected on the shell surface after 28 days' incubation.

Payne (1949) demonstrated the effect of humidity on the survival of *Bact. coli* on the skin of the hand and concluded that the death rate depended on the rate of drying. Maximum death rate occurred at relative humidities of between 40 per cent and 50 per cent. De Ome (1944) studied the death rate of *S. pullorum* when dispersed in a dust-free atmosphere and found that at a constant temperature, the death rate increased with increase of relative humidity.

The present observations were made during preliminary work on the fumigation of egg incubators and one of the main reasons was to determine the limitations of the experimental methods for the evaluation of incubator fumigants. Artifically contaminated eggs, debris, etc., are necessary for the examination of the efficiency of various disinfectant procedures.

Methods

Clean uncracked newly laid eggs were contaminated with *S. pullorum* in one of the following ways:—

(1) The eggs were immersed for a few seconds in a saline or distilled water

suspension of *S. pullorum* prepared by adding 20 c.c. of an overnight broth culture to 150 c.c. of physiological saline or distilled water. This dilution gave a viable count of approximately 60 million organisms per c.c.

(2) The eggs were immersed in a similar saline suspension containing in addition 15 gm. autoclaved hen fæces.

(3) The eggs were treated either by dropping 0.04 c.c. of undiluted broth culture on to the surface of the shell; by smearing the shell with undiluted broth culture or with a suspension of liver from a *S. pullorum* infected chick. Over this latter inoculum was smeared a small amount of fresh unautoclaved hen fæces.

(4) The eggs were smeared with a broth suspension prepared from the liver of a *S. pullorum* infective chick. These eggs were not smeared with fæces.

After treatment by one of the foregoing methods the eggs were placed on wire incubator trays or on unused papier mâché egg trays. They were divided into two groups, one of which was placed in a forced draft incubator of standard design, and the other was left at room temperature but was not protected from the usual small air currents in the room.

Two forced draft incubators of different manufacture were available for the work and these were operated at the optimum temperature and humidity required for the incubation of hens' eggs, namely 100° F. dry bulb, and 85° F. wet bulb. In all experiments the incubator fans were left running and the incubator ventilators were open.

After the eggs had been exposed to the above conditions for the required time, they were transferred by means of sterile forceps to screw-capped wide-mouthed jars containing 200 c.c. of selenite F. medium. The jars were incubated at 37° C. for 40 hours and a loopful streaked on a quarter of a dry desoxycholate citrate agar plate, containing 1 per cent sucrose and 1 per cent salicin. These plates were incubated for 48 hours and the results recorded. All non-lactose fermenting colonies were examined by the rapid slide agglutination test using the appropriate Salmonella antiserum.

Results

(1) *Eggs dipped in a saline or distilled water suspension of S. pullorum.*

For the first part of this work a laboratory strain of *S. pullorum* (No. 364) was used. Results indicated that there was no appreciable difference in the survival time of a suspension of *S. pullorum* prepared by either of the foregoing methods. In each experiment, the comparison between the two types of suspensions was made in the same incubator at the same time. These results also showed that there was a rapid disappearance of *S. pullorum* from the surface of an egg during the course of 24 hours' incubation. In 12 separate experiments, 17 of 71 eggs dipped in a saline suspension yielded viable *S. pullorum*. The corresponding figures for the distilled water group were 14 infected eggs out of a total of 70.

Experiments were made to study the rate of disappearance of *S. pullorum* from both incubated and non-incubated eggs. It will be seen from Table 1

that after 24 hours' incubation, *S. pullorum* had disappeared from 50 out of 54 eggs (92.5 per cent); whilst similar eggs left at room temperature for the same period of time gave 89 per cent recovery for *S. pullorum*. The absence of *S. pullorum* from contaminated eggs incubated for 48 hours is in marked contrast to similar eggs left at room temperature. There was no difference in the survival time of contaminated eggs placed in either of the two forced draft incubators used in this study.

TABLE 1

Survival of S. pullorum on the surface of the hen's egg

Time of exposure of contaminated eggs (in hours)	Survival rate of eggs placed in incubators	No. of observations	Survival rate of eggs left at room temperature	No. of observations
0	—	—	18/18	3
2	11/17	2	6/6	1
4	7/18	2	—	—
6	9/24	3	12/12	2
8-10	9/18	2	6/6	1
24	4/54	5	16/18	3
48	1/36	4	26/54	6
67	—	—	2/5	1

The numerator=the number of eggs infected with *S. pullorum* at the end of the period of exposure; the denominator=the number of eggs used.

(2) *Eggs contaminated with S. pullorum in the manner indicated under methods 2, 3 and 4.*

These experiments were undertaken to try to determine some of the factors which may have influenced the results previously obtained. The results have been summarised in Table 2 and show that the various methods used for the contamination of the egg shell did not alter materially the rate of disappearance of *S. pullorum* from the surface of the hen's egg.

TABLE 2

Survival of S. pullorum after contamination of the egg by various methods.

Method of contamination	Time of exposure in incubator		Time of exposure at room temperature	
	24 hours	48 hours	24 hours	48 hours
Method No. 2 (with fæces) ...	2/6	0/6	6/6	6/6
Method No. 3 (with fæces) ...	2/30	—	12/12	—
Method No. 4 (with natural material)	5/24	0/18	22/23	16/18
Totals	9/60	0/24	40/41	22/24

Numerator=eggs infected; denominator=eggs examined.

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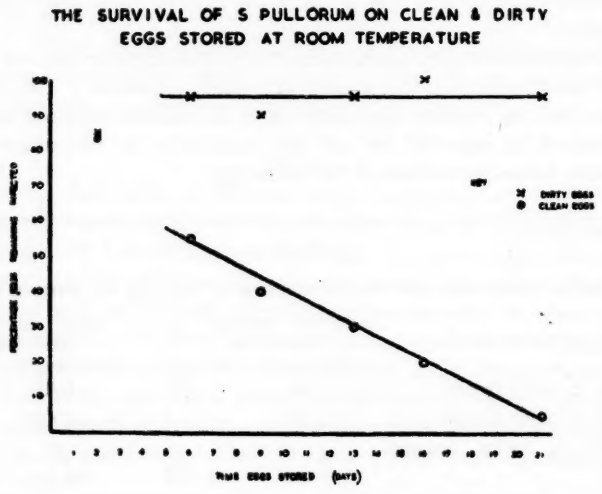
(3) *Eggs contaminated with different strains of S. pullorum.*

Eggs in groups of 16 were dipped in saline suspensions prepared separately from five stock strains of *S. pullorum* and from the second subculture of a newly isolated strain (No. 253). The contaminated eggs were incubated for 24 hours and cultures prepared and the results show that there was no difference in the viability of the strains used. All eggs cultured at 0 hours were positive for each strain and after 24 hours incubation one positive egg was obtained from each of two strains; the remaining 60 eggs yielded no viable *S. pullorum.*

(4) *The survival of S. pullorum on small pieces of incubator debris.*

Dry incubator debris, which included chick down, small pieces of egg shell and dust was mixed with a distilled water, a saline or a peptone broth suspension of *S. pullorum.* Small amounts of this debris (each about the size of a pea) were placed on incubator trays which were transferred to the incubator or left at room temperature. Pieces of material were removed at intervals and cultured in selenite broth. All pieces of debris remained infected with *S. pullorum* after 24 hours in the incubator but not for 48 hours. Survival time of *S. pullorum* in incubator dust at room temperature was not determined.

Fresh (non-sterile) pieces of egg shell when dipped in a saline suspension of *S. pullorum* remained infected for 30 hours in the incubator but not for 48 hours. This contrasted with later experiments using pieces of autoclaved egg shell stripped of the membrane. Similar pieces of contaminated egg shell were left at room temperature and remained infected for at least 48 hours.



(5) *The survival of S. pullorum on clean and dirty eggs stored at room temperature.*

One hundred and twenty clean eggs were dipped in a saline suspension of *S. pullorum* (Strain No. 364). An equal number of eggs were dipped in a similar suspension to which was added 20 grams fresh hen fæces for every 170 c.c. suspension. After draining for one hour, both groups of eggs were stacked on papier mâché trays and stored at room temperature. Batches of 20 eggs were examined from both the clean and dirty groups at intervals by flaming a small area at the large end of each egg, with burning spirit, and transferring approximately 5 c.c. of the contents to a tube of selenite F. medium. The remainder of each egg, including the shell, was then placed in a screw-capped culture jar containing 200 c.c. selenite medium.

It will be seen from the above graph that after 21 days at room temperature it was not possible to recover *S. pullorum* from the surface of most of the clean eggs, whereas the dirty eggs continued to yield *S. pullorum* in large numbers at the end of the three-week period. *S. pullorum* was not isolated from the contents of any egg and there was no evidence of bacterial penetration during the period.

Salmonella thompson and typhi-murium

Methods

(1) Single, clean uncracked newly-laid eggs were placed in sterile wide-mouthed screw-capped jars and inoculated with two drops (total 1/25 ml.) or four drops of broth culture of *S. pullorum, S. thompson* or *S. typhi-murium*. The cap was replaced on the jar and the eggs left at room temperature, for one or five hours, after which about half the jars were transferred to either of the forced draft incubators.

After exposure to room or incubator conditions a sufficient quantity of selenite F. medium was poured into each jar, the jars were incubated for 48 hours and subsequently plated.

(2) Eggs were immersed for a few seconds in a saline suspension of *S. thompson and S. typhi-murium* prepared by adding 200 c.c. of an overnight broth culture to 150 c.c. of physiological saline. In some experiments, eggs inoculated by immersion were allowed to drain at room temperature and later transferred to the egg incubator.

A small hole was drilled in the shell in an area which had been flamed and 2 ml. of egg white was transferred to a tube of selenite F. medium. Preliminary experiments had shown that 1/50 ml. of undiluted broth culture of the three Salmonellæ used in this work, when added to 9 ml. of undiluted egg white and subsequently incubated gave rapid growth as judged by subsequent plating. This ability to grow in egg albumen was confirmed by the inoculation of 3/50 ml. of undiluted culture into the white through a hole which was then sealed with wax; the egg was incubated and later cultured.

Results

Table 3 indicates that under the conditions employed there was no marked

difference between the survival time of *S. pullorum, S. thompson* and *S. typhi-murium,* following inoculation for 25 hours.

TABLE 3

Survival of Salmonella on eggs contained in screw-capped jars.
(25 hours incubation at 37° C.)

S. pullorum		*S. thompson*		*S. typhi-murium*	
Incubator	At room temperature	Incubator	At room temperature	Incubator	At room temperature
13/50	36/44	7/40	37/40	4/34	28/34

Nominator=positive eggs; denominator=number examined.

A study of the survival of these Salmonellæ under other conditions was made and fertile eggs contaminated by immersion in a suspension of *S. thompson* were placed with non-inoculated fertile eggs in a small "still air" drawer incubator; similar inoculated eggs were kept at room temperature in screw-capped jars. Groups of six eggs were cultured at intervals of four days. With the exception of one on the 4th day and one on the 16th, all incubated contaminated eggs failed to yield *S. thompson.* None of the non-inoculated eggs yielded *S. thompson,* neither did the contents of all the eggs. No Salmonellæ were recovered from a number of chicks which hatched. The eggs left in jars at room temperature showed a gradual disappearance of *S. thompson* although three out of six eggs yielded growth after 33 days. There was no indication of penetration of the egg shell during this storage at room temperature.

The above experiment was repeated with *S. pullorum, S. thompson* and *S. typhi-murium* in a forced draft incubator. Groups of 10 eggs and egg contents were cultured at intervals.

Apart from one positive *S. thompson* egg on the 4th day, all eggs and egg contents yielded no growth of Salmonella. A number of chicks hatched, which appeared healthy and these were killed at 14 days and cultures made; no Salmonellæ was isolated. The shell and contents of six infertile and "dead-in-shell" from each of the three groups were cultured after 23 days' incubation. *S. typhi-murium* was recovered from two eggs, *S. thompson* from one; *S. pullorum* was not recovered.

In a similar experiment with eggs cleaned with wire wool before immersion in *S. pullorum* and *S. thompson* suspension, the shell was not penetrated; cultures of egg contents were negative after 14 days' incubation in a forced draft incubator. *S. thompson* and *S. pullorum* disappeared from the surface of the eggs after 14 days' incubation, whilst similar eggs kept at room temperature in jars, were positive at 14 days.

The effect of increased CO_2 tension and humidity on the survival rate of S. pullorum and S. thompson.

Eggs were placed in screw-capped jars and contaminated (Method 1). Carbon dioxide was added to each jar to give final concentrations of 0.5 per cent and 1.0 per cent, and the eggs incubated in these atmospheres for 48 hours.

There was no difference in the survival time of *S. pullorum* and *S. thompson* compared with control eggs in jars with normal atmosphere.

The humidity in jars containing *S. pullorum* or *S. thompson* contaminated eggs was increased by adding 2 c.c. distilled water to each jar before sealing. Groups of six eggs were cultured at intervals and the results are shown in Table 4; these results are in marked contrast with those in Table 3.

TABLE 4

Survival on clean eggs placed in sealed jars with increased humidity.

(Temperature 100° F.)

Time of incubation	*S. pullorum*	*S. thompson*
1 day	6/6	6/6
2 days	5/6	6/6
4 days	6/6	6/6
8 days	4/6	3/6
12 days	1/6	0/6

Nominator=number of eggs yielding the organism; denominator=number examined.

In this experiment, the contents of each egg was cultured and the test organism was recovered from the contents of two of 24 *S. pullorum* contaminated eggs and from a similar number of the *S. thompson* contaminated eggs.

As with the experiments using *S. pullorum,* penetration of the egg shell after external contamination with *S. thompson* was infrequent. In one series, 38 eggs were smeared with a culture of *S. thompson* and incubated for 21 days; cultural examination of the egg contents gave no Salmonellæ. In another experiment, 96 eggs were dipped in a broth culture of *S. thompson;* half of these eggs had been kept previously at room temperature and the others had been incubated at 100° F. for three hours before contamination. All eggs were then placed in a small still-air drawer type incubator. The contents of 60 eggs were cultured at intervals during incubation and all failed to yield Salmonellæ; three out of 24 clear and " dead-in-shell " yielded *S. thompson.*

Discussion

There is little doubt that under the methods described, *S. pullorum, S. thompson* and *S. typhi-murium* do not live on the surface of the hen's egg for any appreciable length of time, in a still-air " drawer " incubator or in the large forced draft machine. This is in marked contrast to the survival time on similar eggs kept at room temperature. There were slight variations in the results and these must be partly due to the small numbers of eggs used. Another cause of this small variation was because wet contaminated eggs were placed on the papier mâché egg trays and these were placed directly in the incubator. In many cases a small quantity of the inoculum had drained from the egg to the bottom of the " cone " on the papier mâché tray, whereas, when the eggs were separated by china eggs and packed in a normal manner on wire incubator trays, the results were more consistent.

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A similar survival time occurred with incubator fluff, debris, etc., contaminated with *S. pullorum,* and the contaminated fluff remained damp in the incubator and did not dry to the fine powder-like material found under normal conditions.

The observation of Gwatkin (1926) on the survival of *S. pullorum* on pieces of sterilised egg shell dipped in a bacterial suspension were repeated and confirmed, and *S. pullorum* remained viable for at least 20 days, at 37° C. and at room temperature.

It is unwise to compare the results with pieces of sterilised egg shell and whole fresh eggs, as natural conditions are reproduced with whole eggs.

The mechanism involved in the death of Salmonella is not clear; the type of inoculum does not influence the death rate and removal of the protective "bloom" from the surface of the egg with wire wool did not affect the survival time or penetration of the organism. The inhibitory action of egg albumen did not influence the results as the Salmonella used would grow in undiluted egg white; this agrees with the findings of Buxton and Gordon (1947).

The CO_2 content of an egg incubator running at full capacity might play a part in the survival of Salmonella, this CO_2 content is approximately 0.4 per cent. The alteration of the content to 0.5 per cent and 1.0 per cent CO_2 failed to increase the survival time at incubator temperatures.

S. pullorum and *S. typhi-murium* survived at incubator temperature 8-12 days longer with an increased humidity and it might follow that the disappearance of Salmonella from the surface of the eggs in incubators was due to a drying effect, in spite of the fact that, in all experiments, the incubator wet bulb reading was of the order of 85° F. (53 per cent R.H.). This drying effect may also account for the short survival time at incubator temperature of Salmonella on eggs in screw-capped jars without increased humidity. Gregory (1948) has shown that the survival of *S. typhi-murium* on the shells of turkey eggs may be influenced by the humidity present in the incubator and the present results correspond to these findings and also to those of Payne (1949). De Ome (1944), showed that any protective agent, such as dispersed broth or dust particles, increased the survival of *S. pullorum* in the air at a given temperature and humidity. The present studies, therefore, may not be strictly comparable to the work reported by these authors.

Several authors have shown that *S. pullorum* and *S. thompson* are capable of penetrating hen and duck eggs. (Buxton and Gordon, 1947; Wilson, 1945, 1948.) Gregory (1948), has reported the penetration of *S. typhi-murium* and *S. bredeny* through the shell of chicken and turkey eggs under different conditions of humidity. He recovered *S. typhi-murium* from the contents of three of 100 eggs artificially contaminated with this organism on the surface. *S. typhi-murium* resembles *S. thompson* in regard to penetration because as reported in the present paper, penetration of *S. pullorum* and *S. thompson* into the white of the egg occurred in a very small number of eggs only. Penetration of a small proportion of eggs followed the smearing of culture or liver suspension on the surface of eggs previously kept at 37° C. and subsequently allowed to cool.

This apparent lack of penetration may have been the result of failing to culture the whole of the egg contents (in the majority of experiments only 2 to 5 c.c. of the egg contents were cultured), or to low virulence of the organisms used, (Wilson, 1948, unpublished data). The porosity of the egg shell plays a part and one experiment indicated that there was rapid penetration of Salmonella when the culture was placed on the surface of porous shelled eggs previously kept at 100° F. and subsequently allowed to cool. This penetration was no doubt mainly mechanical and due to contraction of the egg contents during cooling. This mechanical penetration would be a likely occurrence under natural conditions when the egg may become contaminated with Salmonella during its passage from the cloaca or from dirty nest boxes.

The difference in survival rate of *S. pullorum* contaminated clean and dirty eggs stored at room temperature (Graph 1) is interesting and extends the room temperature results shown in Table 2. It appears that the fæces exerted a protective action on *S. pullorum* which was not apparent in some of the earlier experiments at incubator temperature (Table 2). In later experiments, using naturally dirty eggs (in contrast with the previously used artificially dirty eggs), *S. pullorum* and *S. thompson* survived on a small percentage of eggs for 12 days in a forced draft incubator. It is partly this protection of the Salmonella by dried mud, fæces, debris, fluff, etc., that would indicate the continued need for disinfectant measures for the control of egg-borne infections. On the other hand, should the findings given in this report be confirmed, it would indicate that the external contamination of the shell of a naturally clean egg with *S. pullorum* and *S. thompson,* is not a frequent method of transmitting these infections in a forced draft incubator, where hatching takes place once weekly, or in cases where a separate hatcher is used. This is not in agreement with the field evidence reported by Wilson (1945), that the setting of eggs contaminated with *S. thompson* was a means of spreading the infection within an incubator. However, the present findings, in respect of *S. pullorum,* give experimental confirmation of the statement of Wilson (1948) that contamination of the outside of the shell with *S. pullorum* probably plays little part in the spread of the disease.

Summary

(1) A study has been made of the behaviour of *S. pullorum, S. thompson* and *S. typhi-murium* when placed on the surface of the hen's egg. The results show that these Salmonella disappear rapidly from the shell of the egg under normal egg-incubator conditions. At room temperature the organisms survive for approximately 21 days on clean eggs and for a longer period on artificially dirty eggs.

(2) Experiments with atmospheres of increased humidity suggest that the rapid death of the Salmonella under incubator conditions might be due primarily to a drying effect.

(3) Under the conditions described, penetration of *S. pullorum* and *S. thompson* into the white of the egg occurred a very small number of times.

(4) External contamination of the shell of a naturally clean egg with *S.*

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pullorum and *S. thompson* would therefore appear to be an unlikely means of introducing infection when a separate hatching compartment is in use.

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Acknowledgments

The authors wish to express their gratitude to Dr. R. F. Gordon for his constant interest and guidance during the course of this work and Mr. C. G. Stacey for the supply of experimental material.

STUDIES ON THE RELATIONSHIP OF LUNGWORM INFESTATION IN CATTLE AND THEIR LIVER VITAMIN A RESERVES

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Since the discovery of the vitamins at the beginning of this century, a great amount of knowledge has accumulated regarding their importance in health and disease. Whilst many of them have well-known specific effects, especially relative to the growth of certain tissues or to growth in general, their relationship to the phenomena of resistance and immunity to disease is indirect. It has been shown, however, that individual vitamins have a rôle to play in the body's defences against parasitic infestations. Watt (1944) recovered more *Nippostrongylus muris* from the intestines of rats fed, before infection with larvæ of this parasite, for 56 days on a diet partially deficient in vitamin B_1 (thiamine). The reverse of this was recorded for the control rats fed a basal complete diet. Similar results were recorded in cases of rats fed a vitamin B_2 (riboflavin) deficient diet. Serum of rats fed a diet deficient in B_1 and B_2 proved ineffective in the transmission of passive immunization.

The importance of vitamin A in maintaining the normal physiological activity of the mucous membranes of the body is generally accepted. For many years it has been described as the anti-infective vitamin, though this effect is probably indirect and dependent upon the ability of the vitamin to maintain the epithelial surfaces in a sound vigorous state. A deficiency of this vitamin has been linked (Siefried [1930], Green and Mellanby [1928], Mellanby

[1929], Green Pinder, Davis and Mellanby [1931]), with certain septic lesions in the respiratory and urinary tracts.

Of relationship between vitamin A and resistance to parasitic infestations, it appears that Woodland (1924) was the first to observe that green food in the diet rendered mice refractory to infestation with *Hymenolepis nana* var. *Fraterna.* He stressed the lack of vitamin B and not A as the latter occurs widely as carotene in green food

Hiraishi (1927, 1928) published some observations on *Ascaris* infection in swine in the absence of vitamin A. He infected vitamin A deficient pigs with a human strain of *Ascaris lumbricoides* and produced heavier infestations with a swine strain in deficient pigs. Controlling all other factors he concluded that vitamin A had a direct bearing on the susceptibility of the pig to roundworm infestation.

Ackert, McIlvaine and Crawford (1931) in America produced evidence of the effect of vitamin A on infection in the chicken with *Ascaridia galli.* Larger numbers and longer parasites were obtained from vitamin A deficient chickens.

Nagoya (1931a, 1931b), in Japan, experimenting with larvæ of *Ancylostoma caninum* in dogs, showed that the vitamin A content of the diet affected the percentage mortality of the larvæ when given per os or through the skin.

Foster and Cort (1931, 1932), experimenting with *Ancylostoma caninum* were able to demonstrate the lowering of vitality and resistance when vitamin A was deficient. They were able also to establish partial immunity and partial cure by regulating the vitamin A content of the diet. Their conclusions are somewhat weakened by the fact that the diet not only was deficient in the vitamin but also in mineral salts.

Spindler (1933) found that vitamin A deficient rats showed lowered resistance to superinfection with *Nippostrongylus muris,* whereas normal rats displayed a marked resistance to superinfection with the same parasite.

Clapham (1934) carried out experiments on the lines reported by Hiraishi (1927, 1928) dealing with the effect of avitaminosis A in pigs to *Ascaris* infection. From the results obtained she was unable to come to such a definite conclusion as Hiraishi.

McCoy (1934), experimenting with *Trichinella spiralis,* observed increased persistance of worms in the intestines of rats when they were kept 2 to 3 weeks on a vitamin A deficient diet. More larvæ were observed in the muscles of the deficiently fed than in the adequately fed control animals and a second infection produced no immunity.

Watt (1934) reduced the resistance of the rat to *Nippostrongylus muris* by keeping it on a diet deficient in vitamin A.

It seems that information in the literature on the relationship of vitamin A and lungworm infestation is very scanty. The only reference traceable is the work of Shaw (1937) on the effect of vitamin A deficiency on lungworm infestation in sheep. He came to the conclusion that wethers fed on vitamin A deficient diet for 19 months did not show increased susceptibility to lungworm infestation

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and developed urinary calculi composed mainly of silica. All the experimental wethers exhibited symptoms of A avitaminosis on the diet designed in the experiment for this purpose.

Purpose of the Work

In 1950, during the summer and later on, investigations were made to provide preliminary information about the relationship of vitamin A to lungworm infestations in cattle, and these comprised records of vitamin A levels from livers of cattle that died or were killed as a result of parasitic bronchitis. The liver level of vitamin A in cattle is not a constant figure for, clearly, it might be expected to vary within a wide range according to variations in breed and age, different ways of management and time of the year [Braun (1945), Wise, Atkeson, Caldwell, Parrish and Hughes (1947), Stewart, McCallum and Watt (1952)]. Normal controls were not available, nevertheless, it was considered that if enough material was to hand, the data collected might shed some light on this problem.

Material and Technique

The material comprised liver samples from cattle that died or were killed as a result of parasitic bronchitis. The total weight of each liver and the parasitological and necropsy findings were recorded. Two samples of adult *D. viviparus* were collected from two cows, killed *in extremis,* and the vitamin A contents in the parasites were recorded. Liver samples from guinea-pigs experimentally infected with *D. viviparus* and *D. filaria* were also among the material collected in this investigation.

As Sen and Sharma (1936) and others stated that the central portion of the liver contains more vitamin than in the periphery, samples were removed with scissors from different parts of this organ to make a total of 5 grams. This amount was digested in 10 ml. of 5 per cent aqueous solution of potassium hydroxide at 80° C. and, after cooling 5 c.c. absolute alcohol were added as a preservative. The vitamin A content was assayed by the Carr-Price method as modified by Davies (1933), and the final readings were taken using the photo-electric spectro-photometer described by Thompson (1949). The two samples of adult parasites were similarly treated.

Results

Certain details from 31 animals are recorded in Table No. I.

In this investigation the carotene level in the livers was not recorded; only the vitamin A reserves were estimated. According to Braun (1945) the first demand on liver reserves affects the carotene without decreasing the vitamin A level, but Stewart and McCallum (1952) showed that the levels of both carotene and vitamin A were uniformly affected. Whatever the true state of affairs may be, it was felt that the vitamin A concentration in the liver without the carotene figure provided a valuable index.

The mean vitamin reserve for the 31 cases, taken collectively, is 52.6 I.U./gm. of liver while that for the 23 cows, 3 years old and over, is 66 I.U./gm. of liver, and that for the eight animals, one year old and under, is 14 I.U./gm. of liver.

TABLE I.

Vitamin A concentration in liver samples of cattle affected with parasitic bronchitis.

Case No.	Breed	Age in Years and Months	Termination	Date	Vit. A. I.U./gm.	Duration of Clinical Symptoms	No. of Worms in Lungs	Intestinal Parasite Egg Count	Post-mortem Findings			
									Bronchitis	Pneumonia	Emphysema	Cavity Formation
1	A	3 —	K	9. 8.50	46.3	10 days and in calf	2,750	Nil	+	+++	++++	—
2	A	3 —	K	9. 8.50	135	10 days and in calf	1,425	Nil	+	++	++++	++
3	A	3 —	D	10. 8.50	65.7	10 days	3,910	Nil	—	+	+++	—
4	G	— 8	D	12. 8.50	42.3	14 days	127	4,500/g	+	++	—	—
5	A	3 —	D	12. 8.50	20	10 days and in calf	672	Nil	—	+	+++	+
6	A	3 —	K	12. 8.50	79	9 days and in calf	778	Nil	—	++	+++	—
7	A	4 —	K	12. 8.50	160	12 days	935	200/g	—	—	+++	—
8	A	—10	D	15. 8.50	18	14 days	380	35,000/g	—	++	+	—
9	A	3 —	K	18. 8.50	5	18 days and in calf	1,118	Nil	—	++	+++	++
10	A	3 6	D	21. 8.50	9	21 days and in calf	3,214	Nil	+	++	+++	—
11	S	— 8	K	23. 8.50	13.9	10 days	450	2,800/g	—	++	—	—
12	G	— 6	D	23. 8.50	3	14 days	910	800/g	+	++	—	—
13	S	1 —	D	23. 8.50	1	30 days	1,220	800/g	++	++	—	—
14	A	3 —	D	29. 8.50	8	18 days and in calf	6,150	Nil	—	++	+++	++
15	A	— 8	D	8. 9.50	3	10 days	1,127	2,500/g	—	++	—	—
16	J	4 —	D	12. 9.50	91	30 days, aborted	815	600/g	+	++	++	—
17	A	3 —	K	18. 9.50	102	14 days and in calf	462	Nil	—	++	+++	++
18	A	3 6	D	20. 9.50	153	16 days and in calf	1,260	Nil	—	+	++	—
19	A	3 —	K	22. 9.50	138	21 days and in calf	1,690	Nil	—	+	+++	++
20	G	— 8	K	24. 9.50	16.6	10 days	241	3,100/g	+	+	++	—
21	A	3 —	D	26. 9.50	79	28 days and calved	262	Nil	—	+	+++	—
22	A	3 —	K	26. 9.50	141	16 days and in calf	773	Nil	—	+	+++	—
23	A	3 6	K	10.10.50	11	55 days, recovered	279	Nil	—	—	++	—
24	A	3 6	K	10.10.50	5	52 days, recovered	195	Nil	—	—	++	—
25	A	3 —	K	11.10.50	34	50 days, recovered	889	Nil	—	Collapsed areas	++	++
26	A	3 —	K	11.10.50	54	50 days, recovered	312	Nil	—	—	++	—
27	A	3 —	K	12.10.50	5	30 days and in calf	645	Nil	+	++	+++	++
28	A	3 —	K	17.10.50	87	40 days and in calf	298	Nil	—	++	+++	—
29	A	3 —	D	17.10.50	61	40 days and in calf	399	Nil	—	++	+++	—
30	J	1 —	K	25.10.50	14.9	14 days	583	600/g	—	++	+++	—
31	J	3 —	K	7.11.50	29.1	15 days	327 Immature	Nil	+	—	++++	—

A—Ayrshire G—Guernsey S—Shorthorn J—Jersey K—Killed D—Died

As previously mentioned normal control cattle were not available for comparison. Moreover, the cases recorded were from scattered outbreaks of parasitic bronchitis in different localities of Southern England. The results obtained were compared with the normal levels recorded by Stewart and McCallum (1952) whose survey revealed a seasonal variation for these values in normal cows. The lowest figures they obtained occurred during April and May and the highest during November and December. The average normal value for April over four years was 99.2 I.U./gm. and that for November 1945 and 1948 was 404 I.U./gm. of liver. Although the figures given for normal animals by Stewart and McCallum included both carotene and vitamin A whereas the figures in this study refer only to the vitamin A level, it is nevertheless apparent that there is a severe depletion of the vitamin A reserves in all cases with parasitic bronchitis.

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The Vitamin A content of Adult D. viviparus

To find out if the nematodes in the lungs of these cattle contained any vitamin A reserves in their bodies, two lots of freshly mature *D. viviparus* from two different cases were dried between filter paper, after being washed in normal saline, and 5 gm. of each were secured. This amount from each lot was treated similarly to the liver samples. The vitamin A contents were found to be 0.1 and 0.9 I.U./gm. of worms respectively.

Records of Vitamin A reserves in livers of guinea-pigs experimentally infected with D. viviparus and D. filaria

During a study of the parasitic phase of the life-cycle of *D. viviparus* and *D. filaria* in experimentally infected guinea-pigs made in the summer of 1951, the vitamin A liver reserves of some of these animals were recorded. The results are given in Table II.

TABLE II.

Vitamin A levels in livers of guinea-pigs experimentally infected with D. viviparus and D. filaria.

Guinea-pig No.	Interval between start of experiment and autopsy	Vit. A. I.U./gm. of liver	Parasites in lungs	Post-mortem findings
D. filaria infection				
1	22 days	3	18 young adults 1-17 mm. long	Pneumonic areas of lungs
Control ...	22 days	27	Nil	Normal
2	24 days	2	13 young adults 1-23 mm. long	Pneumonic areas of lungs
Control ...	24 days	19	Nil	Normal
D. viviparus infection				
1	5 days	3	Larvæ in third active stage	Lungs slightly congested
Control ...	5 days	4	Nil	Normal
2	15 days	1	23 young adults 1-2 mm. long	Pneumonic areas at fringe of lobules
Control ...	15 days	5	Nil	Normal
3	20 days	1	8 young adults 1-8 mm. long	Pneumonia more advanced
Control ...	20 days	3	Nil	Normal
4	35 days	1	6 young adults 2-10 mm. long	Pneumonic areas in both diaphragmatic lobes
Control ...	35 days	8	Nil	Normal

All the animals used were initially 6-7 weeks old and each experimental animal was fed 7,000 lungworm larva, control animals receiving none.

All the animals were nearly of the same weight and kept under the same hygienic conditions, receiving the same diet throughout the experiment.

From these results it is clear that the infected guinea-pigs showed a distinct drop of vitamin A reserves in their livers as compared with the non-infected controls and they support the findings recorded in Table I for naturally infected cattle.

As all the cattle cases herein recorded were affected in the late summer and since according to Stewart *et al.* (1952), their liver reserves of vitamin A in the normal state before infection should be high, it could not be assumed that the infection had been acquired as a result of a vitamin A deficiency. The possible effect of a low level or a complete deficiency of this vitamin on the susceptibility to infection still remains to be studied. It would seem reasonable to suggest, from the observations made, that the depletion of vitamin A is brought about by the extra demands on it created by the destruction of the epithelial lining associated with this parasitic infection and the need for regeneration. The regeneration of the shredded epithelial cells appears to place a heavy demand on the vitamin A reserves of the liver and thus to create a serious reduction in that organ. Adult *D. viviparus* contained little vitamin A in their bodies as the data show and more investigation is needed to discover their vitamin A requirements.

Summary

The vitamin A levels in livers of 31 cattle affected with parasitic bronchitis during the late summer of 1950 were assayed. These, when compared with the findings of Stewart *et al.* (1952) on the normal levels, showed a distinct fall in the liver reserves. Similar results were obtained from guinea-pigs experimentally infected with *D. filaria* and *D. viviparus* when compared with non-infected controls. It could be stipulated that, in cases recovering from secondary broncho-pneumonia, a supply of vitamin A may be therapeutically beneficial.

Acknowledgment

Thanks are due to Dr. S. Y. Thompson of the National Institute for Research in Dairying, Shinfield, for carrying out the vitamin A estimation, and to Dr. N. S. Barron for helpful criticism.

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THE ADMINISTRATION OF METHYL THIOURACIL ("OBESIL") TO FATTENING PIGS

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Introduction

THE literature relating to the administration of antithyroid substances to growing or fattening pigs was comprehensively reviewed by Blaxter, Reineke, Crampton and Petersen (1949), while Robinson and Coey (1951) have dealt with subsequent publications. A detailed summary of earlier observations is therefore superfluous in the present paper. So far as Great Britain and Northern Ireland are concerned, there have been only two published records, apart from our own preliminary note dealing with the present experiments (Worden and Bunyan, 1952 *a*).

Braude and Cotchin (1949) carried out three series of experiments in which methyl thiouracil was fed to pigs of varying initial age, in addition to a fourth series in which thiourea was administered. In the case of methyl thiouracil, results with pigs subjected to long periods of administration were disappointing, and there was a tendency for the treated animals to become shorter, rounder, fatter, and lower on their legs than the control pigs. In one case a pig changed from the bacon to the lard type of conformation. In the case of pigs of a mean initial weight of 170 lb., over an experimental period of five weeks, there was a slight improvement in the food conversion ratio as compared with that for control pigs, but appetite and growth were adversely affected.

Robinson and Coey (1951) administered thiouracil to fattening Large White pigs under different systems of feeding. When individually-fed pigs received 0.1 per cent thiouracil in the meal as from 150 lb. liveweight, they attained weights of 210 lb. almost a week sooner than control pigs, and with a saving of some 45 lb. meal a head, or 20 per cent of the total amount fed to the controls. The incorporation of 0.1 per cent thiouracil in the meal of pigs as from 100 lb. liveweight lengthened the time taken to attain 210 lb. liveweight, but there was nevertheless a saving of some 75 lb. meal (equivalent to 17 per cent) a head by comparison with control pigs. There was no evidence in either case that the administration of thiouracil was productive of fatter pigs, while there were no significant differences between the iodine values of the backfat of the thiouracil-fed and control animals. Robinson and Coey refer to the importance of assessing the actual composition of the weight gains made during thiouracil

feeding for, as Callow (1950) has indicated, where an apparent economy in food utilization coincides with lowered growth rates it may reflect merely a greater incorporation of water into the tissues and not a real increase in the efficiency of food conversion. In experiments carried out at Illinois, Terrill, Hamilton, Krider and Carroll (1950) did not find any significant differences between the amounts of lean, fat, bone and skin, or between the chemical composition of the lean or fat, in thiouracil-fed pigs and control pigs that had been pair-fed over a 28-day period prior to slaughter. As Robinson and Coey state, this would suggest that, where the feeding of thiouracil gives rates of liveweight increase not less than those of the controls, the reduced food consumption represents a real caloric advantage.

Vander Noot, Reece and Skelley (1950) found that methyl thiouracil was a more potent goitrogen for pigs than thiouracil, and obtained successful results from its incorporation in the diet at levels of 0.05 and 0.10 per cent. At a level of 0.25 per cent the results were less satisfactory.

We have permission to quote also the records of a farm trial, supervised by a veterinary surgeon, in which methyl thiouracil (as "Obesil," Bayer) was fed to a group of 10 Large White X Essex pigs, with a similar group serving as controls. The trial commenced on 15.9.51, and the groups each received a fixed allowance of 64 lb. meal daily (i.e., 6.4 lb. *per capita*) for the first seven days, and 62 lb. daily (i.e., 6.2 lb. *per capita*) for the succeeding 21 days of the experiment. Thus each group received a total of 1,750 lb. meal, or 175 lb. *per capita*. Dosage with the " Obesil " was carried out according to the maker's current recommendations, viz. 0.5 oz. *per capita* (=1,980 mg. methyl thiouracil) for the first 14 days, and 0.15 oz. (=596 mg. methyl thiouracil) for a further 14 days. The summarised weight records (in lb.) were as follows :—

	Av. initial wt. 15.9.52	*Wt. on* 22.9.52	*Wt. on* 29.9.52	*Wt. on* 6.10.52	*Wt. on* 13.10.52
" Obesil " group	171.8	180.8	189.4	205.8	215.6
Control group	172.8	182.8	193.0	202.2	211.2

The mean gain of the " Obesil " group was therefore 43.8 lb., with a ratio of weight of food consumed to liveweight increase for the period in question of almost exactly 4.0 : 1. The control pigs had a mean gain of 38.4 lb., with a food conversion ratio of 4.56 : 1.

Experimental

The trials were carried out with Large White pigs on the same farm as that at which our recent observations on procaine penicillin were made (Worden and Bunyan, 1952 *b*). The standards of management were excellent, and detailed records of performance were kept. Two comparable groups each of eight pigs were selected, the initial average individual liveweight in each group being approximately 150 lb. During the two weeks prior to the commencement of the experiment the mean weekly individual gain among the animals in each group had been 14 lb. To each animal in one group, 2 g. daily

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of methyl thiouracil (as "Obesil") was given for the first 14 days and 0.5 g. daily thereafter. The second group served as controls. It had originally been the intention to continue the experiment for 28 days only, but owing to deferred slaughtering arrangements it was extended to 35 days. While this delay prevented optimal results from the administration of methyl thiouracil, it provided

TABLE 1—LIVEWEIGHT GAINS (lb.)

(*a*) *"Obesil" group.*

Pig No.	Date of birth	Initial weight, 17.9.51	Weight on 24.9.51	Gain	Weight on 1.10.51	Gain	Weight on 8.10.51	Gain	Weight on 15.10.51	Gain	Final weight, 22.10.51	Gain
1.	15. 2.51	162	180	**18**	204	**24**	220	**16**	234	**14**	240	**6**
2.	15. 2.51	156	174	**18**	189	**15**	202	**13**	210	**8**	212	**2**
3.	17. 2.51	156	178	**22**	196	**18**	206	**10**	220	**14**	220	**0**
4.	24.11.50	154	178	**24**	198	**20**	210	**12**	214	**4**	199	**—15**
5.	26. 3.51	142	155	**13**	175	**20**	193	**18**	202	**9**	214	**12**
6.	12. 2.51	146	171	**25**	190	**19**	208	**18**	217	**9**	226	**9**
7.	3. 1.51	170	193	**23**	210	**17**	226	**16**	238	**12**	243	**5**
8.	22. 2.51	134	160	**26**	176	**16**	190	**14**	204	**14**	212	**8**
	Totals	1,220	1,389	**169**	1,538	**149**	1,655	**117**	1,739	**84**	1,766	**27**
	Average	152.5	173.6	**21.1**	192.3	**18.6**	206.9	**14.6**	217.4	**10.5**	220.8	**3.4**

(*b*) *Control group.*

Pig No.	Date of birth	Initial weight, 17.9.51	Weight on 24.9.51	Gain	Weight on 1.10.51	Gain	Weight on 8.10.51	Gain	Weight on 15.10.51	Gain	Final weight, 22.10.51	Gain
9.	17.2.51	160	182	**22**	198	**16**	214	**16**	218	**4**	222	**4**
10.	17.2.51	160	177	**17**	197	**20**	208	**11**	212	**4**	218	**6**
11.	17.2.51	146	166	**20**	182	**16**	196	**14**	204	**8**	214	**10**
12.	15.2.51	145	162	**17**	180	**18**	198	**18**	202	**4**	226	**24**
13.	15.2.51	150	165	**15**	182	**17**	198	**16**	203	**5**	218	**15**
14.	15.2.51	143	148	**16**	157	**9**	172	**15**	180	**8**	192	**18**
15.	22.2.51	140	157	**17**	173	**16**	186	**13**	180	**—6**	200	**20**
16.	26.3.51	142	151	**9**	176	**25**	192	**16**	198	**6**	216	**18**
	Totals	1,175	1,308	**133**	1,445	**137**	1,564	**119**	1,597	**33**	1,712	**115**
	Average	146.8	163.5	**16.6**	180.6	**17.1**	195.5	**14.8**	199.6	**4.1**	213.8	**14.4**

valuable confirmatory evidence that a period of even four weeks of such administration is probably too long for pigs at this stage of development with the scheme of dosage adopted.

The liveweight gains of the pigs in the two groups are set out in Table 1. These gains may be summarised on a weekly basis as follows (in lb.):—

	"Obesil" group	*Control group*
1st week ...	21.1	16.6
2nd week ...	18.6	17.1
3rd week ...	14.6	14.8
4th week ...	10.5	4.1
5th week ...	3.4	14.4
Total	68.2	67.0

The food consumption of the "Obesil" group during the five weeks of the experiment was 1,456 lb., so that the conversion ratio of food consumed: liveweight gain was the excellent one of 2.67 : 1. The food consumption of the control group was 1,568 lb., with a food conversion ratio of 2.93 : 1. The saving in food in the case of the "Obesil" group was approximately 7 per cent.

On completion of the feeding trial the pigs were all consigned to Messrs. T. Wall & Sons' Bacon Factory, Willesden, where we again had the expert co-operation of Mr. Collan E. Brett and his staff in grading and measuring the carcasses and in securing portions of fat for chemical analysis. The carcass data (see Davidson, Hammond, Swain and Wright, 1936; McMeekan, 1940) are set out in Table 2.

It will be seen that one pig in the "Obesil" group had become overweight, and could not therefore be graded, and that three of the others graded "A," two as "B" and two as "C." Of the control pigs, four graded "A," three as "B" and one as "C." The marks secured were in favour of the control group, which secured a total of 156 against 114 by the "Obesil" group. From the cold carcass weight (W) the estimated thickness (T) of the back fat was calculated from the equation

$$T = 1.40 + 0.149\ W$$

and the difference between the found thickness of back fat (B) and the value T are set out in the table (see Callow, 1942; Worden, 1943). It will be seen that two animals receiving "Obesil" and one of the control animals were significantly fatter than expectation, but that on the whole there was good agreement between the calculated and the found values.

Portions of the inner and outer layers of loin fat were removed from the individual pigs after slaughtering and after pooling their iodine numbers were determined by the method of Wijs (1929). The results were as follows:—

(a) *"Obesil" group*
Inner layer, 63.5,
Outer layer, 65.3.

(b) *Control group*
Inner layer, 62.4,
Outer layer, 61.3.

These data indicate that the fat of the "Obesil"-fed pigs was slightly softer

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TABLE 2—

(*a*) *"Obesil" group.*

Pig No.:	1	2	3	4	5	6	7	8
Carcass weight (lb.)	174	152	162	145	153	163	183	153
Length (mm.)	804	813	789	762	776	813	803	786
Marks for length	2	7	3	4	4	5	1	5
Fore-streak (mm.)	34	28	24	36	37	29	31	34
Mid-streak (mm.)	25	22	29	30	26	25	26	28
Hind-streak (mm.)	26	25	31	29	32	37	35	33
Marks for streak	3	3	4	7	6	5	4	6
Shoulder fat (mm.)	54	47	50	53	1	5	4	8
Mark for shoulder fat	7	8	8	4	61	56	64	47
Shoulder grade	B	A	B	B	C	C	—	A
Loin (mm.)	20	21	24	33	34	35	44	28
Loin grade	A	A	A	B	C	C	—	B
Back fat, B (mm.)	24	25	30	29	35	30	38	28
Back grade	A	A	B	B	C	B	—	A
Total marks	12	18	15	15	11	15	9	19
Final Grade	A	A	B	B	C	C	OW	A
T	27.3	24 05	25.54	23 0	24.2	25.7	28.69	24.2
B—T	—3.3	0.95	4.46	—6.0	10.8	4.3	9.31	3.8

than that of the control pigs, but the difference is only a small one and the iodine numbers may be regarded as satisfactory in both groups (see Lovatt, Worden, Pickup and Brett, 1943). They are a good deal lower than the mean values recorded by Robinson and Coey (1951). As in the case of their animals, the slight but probably statistically insignificant difference between the values for the "Obesil" and the control pigs reflects the difference in back fat thickness.

Discussion

It is clear that antithyroid substances need to be employed with discretion under farm conditions. As Blaxter *et al.* (1949) have stated ". . . modification of thyroid function shows considerable promise for application in swine husbandry. Its proper use, however, will require intelligent consideration of the principles of thyroid physiology involved." The results presented in this paper lend support to those of Robinson and Coey (1951) and indicate that a not inconsiderable saving in food consumption may be achieved through the administration of an antithyroid drug during the last stages—or, rather, during a portion of the last stages—of fattening, and that this saving may be achieved without detriment to final weight or carcass quality. On the basis of our findings further trials with methyl thiouracil have been instituted elsewhere, and the effect of administration for a limited period only, commencing a month from slaughter, is being investigated.

The use of antithyroid drugs, employed with care and precision will probably be found to be economically justified. The indiscriminate use of such compounds can, however, only lead them into disrepute and as yet there does not seem to be any justification for amending the view previously expressed (Worden, 1951) that they should be made available only to the specialist pig farmer. Such drugs for animal use are normally obtainable only through members of the veterinary profession, and it behoves the latter to maintain strict supervision of the animals concerned. It is essential that antithyroid substances should not be administered at an incorrect stage of fattening, nor over a prolonged period.

CARCASS DATA

(b) Control group.

Pig No.:	9	10	11	12	13	14	15	16
Carcass weight (lb.)	167	160	152	164	156	140	147	154
Length (mm.)	831	823	796	813	806	785	802	821
Marks for length	7	6	6	5	7	6	8	8
Fore-streak (mm.)	36	35	31	40	34	32	30	40
Mid-streak (mm.)	37	35	32	34	25	32	26	30
Hind-streak (mm.)	43	26	27	38	30	38	30	37
Marks for streak	9	6	5	8	5	8	5	8
Shoulder fat (mm.)	55	50	55	52	44	45	57	53
Marks for shoulder fat	6	8	4	7	10	7	2	5
Shoulder grade	B	A	C	B	A	A	A	A
Loin (mm.)	35	28	35	35	27	20	22	33
Loin grade	C	A	C	C	A	A	A	C
Back fat, B (mm.)	30	24	35	30	21	20	26	27
Back grade	B	A	C	B	A	A	A	B
Total marks	22	20	15	20	22	21	15	21
Final grade	B	A	C	B	A	A	A	B
T	26.3	28.25	24.05	25.85	24.65	22.26	23.3	24.35
B—T	3.7	—1.25	10.95	4 15	—3.65	—2.26	2.7	2.65

Summary

(1) Details are given of a feeding trial in which a group of eight pigs with a commencing mean individual liveweight of 152.5 lb. were given 2 g. daily of methyl thiouracil (as " Obesil," Bayer) for seven days, followed by 0.5 g. daily for a further 28 days prior to slaughter.

(2) The results indicate that the period of methyl thiouracil administration was too long, but even so there was a saving in food consumption of the order of 7 per cent as compared with a comparable group of eight control pigs, slightly superior liveweight gains and hence a better food conversion ratio. Data on carcass quality and on the iodine numbers of samples of back fat are provided.

(3) The administration of antithyroid drugs to pigs in the later stages of fattening is probably justifiable economically, but their use entails care and precision if satisfactory results are to be obtained.

Acknowledgments

We are grateful to Bayer Products, Ltd., for supplies of " Obesil " and for their support of this work, and to Mr. E. A. Bishop, of Wilts United Dairies, Ltd., and his staff, for the excellent facilities placed willingly at our disposal.

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THE EXPERIMENTAL REPRODUCTION OF SO-CALLED LUMBAR PARALYSIS—EPIZOOTIC CEREBROSPINAL NEMATODIASIS—IN GOATS IN JAPAN

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Introduction

THE disease, which was once commonly known as lumbar paralysis of goats, sheep and horses in Japan and Korea (and goats in Ceylon) has now received good attention in recent British veterinary literature. Innes, Shoho and Pillai (1952), for example, have published a comprehensive account of all aspects of the disease which they contend deserves the pathological designation of epizootic cerebrospinal nematodiasis. Their paper gives the various developments which culminated in the elucidation of the exact nature, and then the helminthic cause now known to be immature *Setaria digitata*. They draw attention to the intensive investigations done by Japanese workers before and during World War II, but which were obscured from sight, partly due to their publication in the Japanese language. A full list of appropriate Japanese references is given by them, thus it is unnecessary to refer to most of the latter. Much stimulus to dissemination of knowledge concerning this nervous disorder of animals, amongst the English-reading public, arose from work by Innes (1951) on a caprine paralysis in Ceylon, for this condition is now known to be identical with the animal paralysis in Japan and Korea. Further, as Innes *et al.* point out, there is inferential evidence that this same disorder has existed in domesticated and wild animals in many other parts of the world, but it has evaded true recognition because of the lack of good pathological studies. In particular they refer to the antiquity of clinical observations on a paraplegia of horses known as Kumri in India, Burma and Assam, which has occurred for at least 100 years. Although complete proof is lacking on the total identity of this disorder, Innes *et al.* think it is cerebrospinal nematodiasis, and that there were shrewd observations before 1900 which pointed to a helminthic cause, although they were never fully investigated. It is necessary to recall only a few cardinal features of the paralytic disease for the purpose of this paper dealing with experimental work.

The disease is an acute, or subacute, epizootic paralysis which occurs seasonally each year in the late summer and autumn. The pathology is described, and with ample illustrations by Innes, Shoho and Pillai, and is fully in harmony

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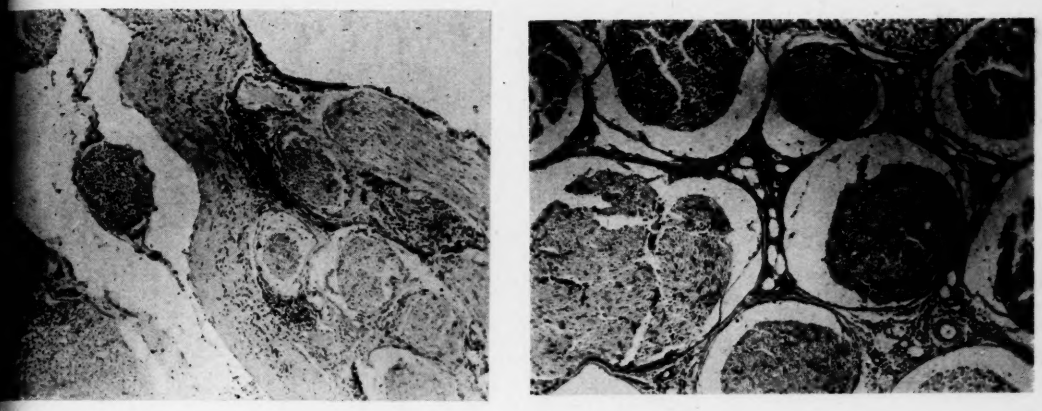

A B

FIG. 1

Goat, No. 2, experimental.—(*a*) *Spinal nerve root,* C-3. Epidural infiltrations mainly by eosinophils, and (*b*) similar intersitial infiltrations in sciatic nerve. (Compart figures of Innes, Shoho and Pillai.)

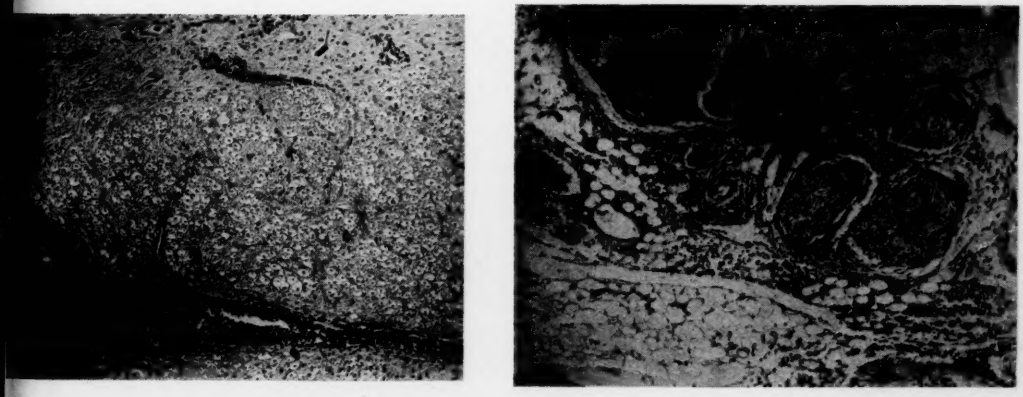

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FIG. 2

Goat No. 4, experimental.—(*a*) *Spinal cord,* C-5. Showing eosinophilic infiltrations along ventral fissure, and some perivascular cuffing in both white and grey matter. (*b*) *Spinal nerve root,* T.3. Showing eosinophilic infiltrations in communicating portion of both dorsal and ventral roots.

(*Article by Ishii, Yajima, Sugawa, Ishiwara, Ogata and Hashiguchi, page* 160)

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FIG. 3

Goat, No. 8, experimental.—Spinal cord, C.3. Malacic lesion with microcavitation and infiltrations extending in from pia, and some evidence of secondary degenerations in the swelling of axis cylinders. (Compare figures of Innes, Shoho and Pillai.)

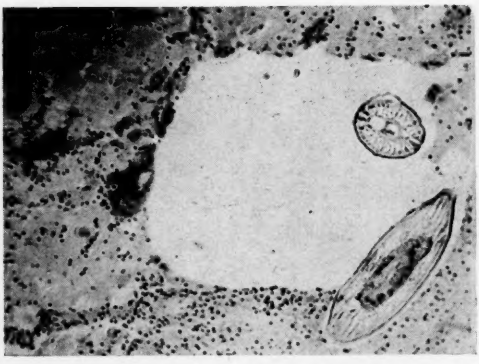

FIG. 4

Goat, No. 5, experimental.—Corpus mamillare, left side. Hæmorrhagic malacic and cavitating lesion with two transverse sections of a single *Setaria digitata,* upper left.

(Article by Ishii, Yajima, Sugawa, Ishiwara, Ogata and Hashiguchi, page 160)

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with Japanese work by Kimura and Niimi (1939-43), Ishii and Sukawa (1944) and Yamagiwa, Shoho and others. The essential lesion is that of a focal encephalomyelomalacia proceeding to liquefaction and cavitation, and there is nearly always a concomitant patchy cerebrospinal meningitis (and even radiculitis of spinal nerve roots) in which eosinophilic infiltrations may predominate. Innes *et al.* (1952) showed that this type of lesion fits with the concept that it is produced traumatically by a wandering worm, and they illustrate almost a replica of the pathological process (minus the eosinophilic infiltration) in the lesions produced by experimental needle stab-wounds of the spinal cord in sheep. Emoto (1927) first described the clinical features and gave brief descriptions of the pathology of the goat paralysis. Later Kimura and Niimi (1939-43), Oguni (1944), Ishii and Sukawa (1944), Yamagiwa and Shoho (1944) and Tanaka *et al.* (1945) made further pathological observations on the caprine, ovine or equine condition, while Taniguchi (1939), Ishii and Watanabe (1942), made some clinical and hæmatogical studies. Many other Japanese workers, too numerous to mention here, also contributed at various stages and these are listed in the Special Korean Research Commission Report (1939-43) or more recently in the paper by Shoho (1951), but unfortunately almost all early papers are in Japanese. The first idea of an helminthic cause came from Fujita (unpublished data); later Kimura and Niimi detected *Setaria digitata* in nervous lesions and they subsequently succeeded, for the first time, in producing the experimental disease in sheep by the subcutaneous injection of infective larvæ of *S. digitata* which had been matured in mosquitoes, *Anopheles hyrcanus sinensis* and *Armigeres obturbans* (*Syn. A. subaltus coquillette*). Many other experiments have been carried out, for example, Itagaki *et al.* (1949) [see Shoho (1951)] reported on some work with horses.

This paper gives details of an experiment carried out in 1951 on goats. The protocols may be of value to English-speaking veterinarians for they indicate the general requirements of such an experiment, designed to amplify observations on the helminthic ætiology of lumbar paralysis.*

Materials and Methods

The experiment was started on June 14 and terminated October 31, 1951. Nineteen goats were used, nine being used for inoculation and the remaining 10 kept as untreated controls. All were kept in a large stable in which the windows and doors were covered by wire gauze to prevent entry of blood-sucking insects. The animals were five months old and weighed about 15 kg. each, and were brought from a table-land on the Japanese main island, where no cases of lumbar paralysis had been reported to occur. Each animal received daily the following fodder ration—0.2 kg. of a mixture of corn, wheat, bran, fish meal, soy-bean, mailo, rice bran, bonemeal; 10 g. NaCl; 10 g. $CaCo_3$; 0.5 kg. oats; 2.0 kg. green grass; and 0.5 kg. hay.

* It is unnecessary to criticise further the contentions of Bush (1951) that there was really no neuro-paralysis of animals in Japan caused by nematodes, but that it was osteomalacia caused by mineral deficiency. This has been done by Innes, Shoho and Pillai, who point out that his conclusions are of little value because of the absence of pathological study.

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A Korean ox, aged three years, was employed as a blood source of microfilaria of *Setaria digitata,* cattle being the natural host of this nematode. The following table (Table 1) indicates microfilarial counts, made during the period when the mosquitoes were allowed to feed on the bovine reservoir of infection.

In an experiment in 1950 *Aëdes togoi* was used as the intermediate insect host, but no successful results were obtained in the case of experimental goats. Itagaki *et al.* (1949), however, has shown that this species of mosquito may be involved in transmission of the disease in young horses. In the experiments described below *Armigeres obturbans* (*Syn. A. subaltus coquillette*) was used, collected from the cesspools of stables and cultured to the adult stage. As soon as the adults showed activity, after about 24 hours' emergence, they were collected in a small cage and allowed to suck blood from the ox between 3-5 p.m.

TABLE I

Number of Microfilaria in the Blood of Cattle

Date of examination	Number of microfilariæ per 20 cmm. of circulating blood	Date of examination	Number of microfilariæ in 20 cmm. of circulating blood
Aug. 2, 1951 ...	9.5	Aug. 14, 1951 ...	19.0
Aug. 3, 1951 ...	6.6	Aug. 16, 1951 ...	15.2
Aug. 4, 1951 ...	17.3	Aug. 17, 1951 ...	11.3
Aug. 6, 1951 ...	16.5	Aug, 22, 1951 ...	20.0
Aug. 7, 1951 ...	15.0	Aug. 27, 1951 ...	14.5
Aug. 8, 1951 ...	14.2	Sept. 4, 1951 ...	10.2
Aug. 10, 1951 ...	12.0	Sept. 5, 1951 ...	6.3
Aug. 13, 1951 ...	10.0	Sept. 10, 1951 ...	7.0

The fully engorged females were then kept in an insectarium and fed artificially on glucose solution, water and green peas. Between 13 and 15 days thereafter they were dissected, infective larvæ isolated from the proboscis and salivary glands and placed in saline solution. They were then injected subcutaneously into the experimental goats in the right side of the neck. The number of larvæ injected and other data are given in Table 2.

Control Animals

No clinical signs of any type of paralytic condition or motor inco-ordination appeared in any of the 10 control goats. At the termination of the experiment five were killed, and a thorough examination was made of the brain and spinal cord; no pathological lesions were found. Histological studies are being made of the bones, the results of which may be reported separately.

Experimental Animals

Seven out of the nine animals showed, at variable periods (14-38 days) after injection of the larvæ, signs or either acute or subacute motor weakness and inco-ordination, which in some animals proceeded to complete paralysis when the animals lay on their side and were unable to stand. In six out of the seven goats part or the whole pathological complex of cerebrospinal nematodiasis

was found in the nervous system, and in widely differing anatomical locations. A summary of the clinical and pathological findings in each experimental goat is given below.

TABLE 2

Results of Injection of Infective Larvæ of Setaria digitata

Goats		Injection of infective larvæ			Results				Pathology			
Number	Age (Months)	Date of Injection	Number	Total Number	Date of onset of the disease	Incubation period (Days)	Duration of experiment before autopsy (Days)	Clinical signs	Macroscopic lesions	Malacia	Meningitis	Parasites present
2	5	Aug. 15 45 " 17 105 " 18 83		233	Sept. 17	33-35	44	+	—	—	+	+
3	5	" 19 183 " 20 123		306	Sept. 3	14-15	20	++	—	+	+	—
8	5	" 21 112 " 21 41 " 22 49		202	Sept. 14	24-25	30	++	+	+	+	—
15	5	" 22 15 " 24 10 " 25 95 " 26 85		205	Oct. 2	38-44	47	+	—	—	+	—
11	5	" 27 80 " 28 80 " 29 37		197	Sept. 25	27-29	101	+	—	—	—	—
26	5	" 29 23 " 30 45		68	No nervous signs		100	—	—	—	—	—
23	5	" 31 38 Sept. 3 13 " 4 54		105	No nervous signs		105	—	—	—	—	—
5	5	" 4 11 " 5 56 " 10 48		115	Oct. 7	28-33	33	++	+	+	+	+
30	5	" 18 6 " 21 19 " 22 23 " 24 55		103	Oct. 10	22-27	55	+	—	+	+	—

Goat 2: Injected with 233 larvæ. After 33 days motor inco-ordination and frequent tumbling, and occasional spasms of the whole body; on the 34th day the animal had difficulty in standing but there was gradual recovery thereafter; the animal was killed for autopsy on the 44th day. *Pathological findings:* No macroscopic changes in brain; a single *Setaria* (1.4 cm. in length) was found free in the subdural space near the lower part of the midbrain; histologically there was patchy eosinophilic meningitis around this region. *Spinal cord:* No naked eye changes; patchy severe eosinophilic meningeal infil-

trations around C.3-C.7, T.1-4, T.10-13 and L.1-6; the same infiltrations were seen in many of the spinal nerve roots, both dorsal and ventral, and also in the proximal part of the sciatic nerves.

Goat 3 : Injected with 306 larvæ. After 14 days hyperacute paralytic signs starting in the hind limbs, proceeding to complete paralysis within 24 hours; non-febrile; no improvement and the animal was killed on the 20th day. *Pathological findings :* No macroscopic changes in brain; occasional areas in periventricular regions of pons and medulla showed perivascular cuffing with eosinophils. *Spinal cord :* Severe meningitis (epidural to pial) with eosinophils and lymphocytes in C.1-7 and T.5-8; the leptomeningeal reaction extended into fissures and septæ of the cord, and was also present in white and grey matter of some levels, in the dorsal and ventral roots of spinal nerves and in the ganglia.

Goat 8 : Injected with 202 larvæ. Acute paralysis developed after 24 days leading to complete helplessness, starting with the hind legs but later involving fore-limbs; non-febrile; killed on the 30th day. *Pathological findings :* A narrow track of hæmorrhagic softening was seen macroscopically, passing from base of the brain through the *corpus striatum* to the lateral ventricle; this was a typical malacic lesion characteristic of the natural disease; eosinophilic infiltration in meninges and the lesion was prominent. *Spinal cord:* Eosinophilic meningitis also involving nerve roots was found at nearly all levels varying in severity, but most marked about C.3, and at this level there was also a malacic focus.

Goat 15 : Injected with 205 larvæ. Motor weakness developed after 38 days, which improved after 4 days, although some impairment of gait was still noted when animal was slaughtered on the 47th day. *Pathological findings :* No changes found macroscopically, but patchy severe eosinophilic meningitis present in cerebral hemispheres, and in the cord around C.4-6, T.2-3 and L.1-3.

Goat 11 : Injected with 197 larvæ. After 28 days slight motor weakness, which persisted for a few days and then slow recovery. Killed on the 101st day. No pathological changes were found in brain or spinal cord.

Goat 26: Injected with 68 larvæ. No clinical signs were noted during the 100 days the animal was kept under observation. At autopsy no pathological changes were found in the brain or cord.

Goat 23 : Injected with 105 larvæ. No clinical nervous signs were seen during the 105 days it was kept under observation. At autopsy no pathological changes were found in the brain or cord.

Goat 5 : Injected with 115 larvæ. After 28 days motor weakness and incoordination with tremors; weakness particularly affecting left hind limb; great difficulty in rising; slow recovery with some residual impairment of gait. Killed after 33 days. *Pathological findings:* A macroscopic lesion was found in the medial part of the left pyriform lobe adjacent to optic tract and anterior part of cerebral peduncle and extending to *nucleus amygdale*; the lesion was about 1 cm. in length and was essentially that of hæmorrhagic softening; a malacic focus typical of the disease was found in histological preparations and in the lesion sections of an immature worm were found. The lesion tracked from the *pia* inwards, and there was a diffuse eosinophilic meningitis, involving nerve roots of the cord—particularly around C.1-5 and T.12-13.

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Goat 30: Injected with 103 larvæ. Nervous signs first appeared on the 22nd day, affecting particularly the right hind limb; when the animal was driven it tended to tumble; slight recovery within a few days; killed after 55 days. *Pathological findings:* No naked eye changes were found in brain or cord; occasional areas of perivascular infiltration in mid-brain, and slight eosinophilic meningitis in C.4 and T.1-3 and in ganglia and nerve roots of these levels.

A few photographs, illustrating some typical lesion of the disease seen in the experimental goats are appended. These should be compared with some of the figures of Innes, Shoho and Pillai (1952).

Discussion

Experiments, of the nature outlined in this paper, are beset with difficulties, are time-consuming and laborious, and meticulous work is necessary at the beginning to provide sufficient infective larvæ for injection. This prevents any experiment being done using a very large number of animals, at least under prevailing conditions in Japan. It should, however, be stressed that this work is only an example of a number of similar experiments which have been made in Japan and Korea and using all the species affected under natural conditions.

A consideration of the known facts concerning the natural history of the disease (see Innes, Shoho and Pillai) will also reveal that there are factors which might influence the final result, i.e., the number of animals in which disease is successfully reproduced. In fact, unless colossal numbers of larvæ were injected, it might be surprising at any time to produce a 100 per cent of positive cases. The cause of the disease rests on the abnormal behaviour of mosquito-stage larvæ of *Setaria digitata* (natural host, cattle) in other domesticated animals as unnatural hosts. Why developing adults *Setaria digitata* do not behave in the same pathogenic way in cattle is a mystery, and what *they may* do in unnatural hosts might be unpredictable. As Innes and Shoho point out, in these circumstances the nervous system may be a strange anatomical tissue of attraction. The disease is usually non-febrile in contrast to neurotropic virus infections. Some animals become affected with an acute paralysis from which there is no recovery, while others are only slightly affected with motor weakness and inco-ordination from which there may be complete remission. Innes and Shoho have pointed out the clinical variations might be reconciled by a consideration of the pathology. For example, it was postulated that in acute cases it is likely there is definite malacic damage to vital motor parts of the nervous system. On the other hand once the worm—as an immature adult—reaches the craniovertebral canal there seems to be no reason for the fact that it may, or may not, penetrate actual nervous substance. Hence, in the latter type one might expect only signs of meningeal and radicular irritation, evinced as inco-ordination during life and by eosinophilic meningitis and radiculitis after autopsy.

It is unknown how many larvæ are introduced by the insect vectors under natural conditions, but clearly it must be a large number. Yet, in all reported cases where worms have been found associated with the disease (and actual

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lesions) only one, two or three at the most seem to finally emerge in the cranio-vertebral canal. Further, in chronic or mild cases, where the animal lingers on for a few weeks no worms may be found at all in nervous system or CSF after a most careful search. In such cases, the presence of an eosinophilic meningitis might be inferred to mean a worm had passed through and wandered to a further unknown destination. All these features are brought by some of our results.

Seven out of the nine experimental goats showed unmistakable clinical evidence of some neurological disorder ranging from motor weakness and ataxia with recovery, to the same condition proceeding to complete neuro-paralysis in a short space of time, and after a latent period ranging from 14-38 days after inoculation of the larvæ. No signs of a neurological disorder were observed in the control goats, kept under the same roof and conditions for a period of over 100 days. The latent period fits in with what is known to occur in the case of animals imported into Japan and Korea during the appropriate season of the year (see Korean Report) and affected naturally. In six out of the seven animals with clinical signs, examination of the nervous system showed the presence of part, or the whole of the pathological complex of cerebrospinal nematodiasis, i.e., a patchy or diffuse eosinophilic meningitis with, or without, a focus of malacia. In consideration of the pathological results our macroscopic examinations were careful and methodical, but the histological examinations were somewhat limited. Innes, Shoho and Pillai have shown it may be necessary to embed the whole brain and length of the cord to obtain a true picture of the process. They admit the task may be formidable, and clearly an experiment involving any big number of animals makes this technique impossible. From this, however, it can be stressed that the lesions described by us in our experimental goats represent only the minimum extent of pathological damage observed.

From the results given in Table 2 it might be deduced that the larger the number of infective larvæ inoculated reduces the latent period of the disease. As under both natural and experimental conditions there are never more than one, two or three worms present in the craniovertebral canal, it must be obvious that most inoculated larvæ die, or in their wandering never reach the nervous system. In Goats 26 and 23 inoculated with 68 and 105 larvæ respectively there was no evidence that worms had ever penetrated the nervous system, either in the form of clinical signs or even mild pathological meningitis. On the other hand Goat 5 was inoculated with 115 larvæ, and the result was an acute flaccid paralysis with slow recovery; a typical malacic focus was found in the brain, and an immature worm was present in the lesion. We do not thus know the train of events from the time of insect (or needle) inoculation of larvæ into an animal leading to acute paralytic disease, to mild signs with recovery, or to no disease at all.

There is no direct proof of the exact path taken by the *Setaria* larvæ from the subcutaneous entry to their final neural destination. Migratory paths have been discussed by Innes and Shoho (1952), and we agree with them that all circumstantial evidence, however, points to a direct tissue-wandering path. It

certainly seems as if vascular paths can be excluded, for as they point out, and prove, the malacic lesion is essentially mechanically traumatic, and is certainly not embolic. The facts that an eosinophilic inflammatory reaction is so frequently seen in meninges, and around the spinal nerve roots in natural cases, and in every single positive experimental goat in this series, would suggest that the developing worms enter the craniovertebral canal *via* the intervertebral foramina along nerve roots. That worms were not identified in each positive case is in harmony with what is known to occur under natural conditions.

In conclusion it is contended that additional experimental evidence has been obtained to show that "lumbar paralysis" in goats is caused by immature worms of *Setaria digitata,* when introduced subcutaneously into unnatural hosts as mosquito-stage infective larvæ.

Summary

(1) Mosquitoes (*Armigeres obturbans—Syn. A. subaltus coquillette*) were raised artificially, and after emergence were fed on an ox carrying the microfilaria of *Setaria digitata* in its blood.

(2) After maturation in insects for about 14 days, infective larvæ were dissected out from the insects, placed in saline and inoculated hypodermically into nine experimental goats and kept in a wire-gauze protected compound along with ten untreated controls.

(3) Seven out of the nine experimental goats after a latent period of 14-38 days exhibited signs of neurological impairment ranging from motor weakness to a following acute paralysis.

(4) Pathological studies demonstrated the presence of part, or whole, of the classical complex of the lesions of cerebrospinal nematodiasis.

(5) The positive results confirm and extend previous experimental work by Japanese workers, concerning the ætiology of the seasonal neuro-paralysis which affects sheep, goats and horses in Japan and Korea (and goats in Ceylon).

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A COMPARATIVE STUDY OF CANINE INTRAVENOUS ANÆSTHESIA IN A TROPICAL COUNTRY

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Introduction

To assess the most suitable canine intravenous anæsthetic for tropical use records have been analysed, and are appended in the table. The following products were used: "Kemithal" Sodium (15 cases); "Pentothal" Sodium (15 cases); "Intraval" Sodium (15 cases) and "Nembutal" Sodium (50 cases). The averages of age, weight, and pre-anæsthetic pulse and respiration rates were common to all cases, and are not included. The main operations performed under each anæsthetic are given to indicate the scope of their use under our conditions. All products were administered at recommended concentrations and speeds, and no deaths occurred.

(1) *"Kemithal" Sodium (Thialbarbitone Sodium) — Imperial Chemical (Pharmaceuticals):*

Urethrotomy, penis amputation, castration, ovariectomy, bone pinning, reduction of dislocation, cataract extraction, splenectomy, and gastrotomy.

In all cases induction was smooth and quiet, with a regular pulse and deep, steady respirations. Three cases showed insignificant salivation which can be controlled by hypodermic injections of atropine sulphate prior to induction. Total induction time was somewhat longer than for "Pentothal" and "Intraval," but considerably shorter than for "Nembutal."

(2) *"Pentothal" Sodium (Thiopentone Sodium)—Abbott Laboratories:*

Ovariectomy, panhysterectomy, and reduction of dislocation. Dyspnœa with accelerated respirations occurred in two cases, and Cheyne-Stokes breathing was common. Dosage was usually half of that for "Kemithal." The time to complete recovery was five times that of "Kemithal" and was accompanied in all cases by "running" and some struggling.

(3) *"Intraval" Sodium (Thiopentone Sodium)—May & Baker:*

Setting of fracture, removal of venereal tumours and tooth extraction. Dosage was two-thirds greater than with "Pentothal," while the time to complete recovery was somewhat shorter. The same "running" complication was seen, with the addition of considerable howling. One case of apnœa occurred and required one minute's artificial respiration. Cheyne-Stokes breathing was seen in nearly all cases, and was alarming in some.

(4) *"Nembutal" Sodium (Pentobarbitone Sodium)—Abbott Laboratories:*

Enucleation of the eye, plastic keratatomy, reduction of dislocation, bone pinning, aural resection, pneumenectomy and lobectomy, thoracic œsophagotomy, thyroidectomy, major abdominal surgery. Dosage was the same as for "Kemithal," though induction time was longer, and was accompanied in

several cases by excitement and howling. Five cases required resuscitation by oxygen during anæsthesia, and several more were given intravenous stimulants. Recovery was attended by the same " running " and " howling " as seen with " Intraval," and averaged 19 hours from induction.

Conclusions

Should the rate of injection, as indicated by the weight of the subject and dose given, of the thiopentone sodium formulations be slightly too fast apnœa is apt to supervene. The margin of error, however, is very small. At correct injection rates Cheyne-Stokes breathing is common, and the accelerated compensation associated with it is inconvenient, especially during abdominal surgery. Abdominal relaxation is rarely as complete as with " Kemithal " and " Nembutal." The " running " on recovery can seriously interfere with dressings, particularly on the limbs, while the additional howling experienced with " Intraval " and " Nembutal " is undesirable in an out-patients' hospital.

" Nembutal," on the other hand, gives a long period of post-operative narcosis, which is useful in reducing interference with dressings in painful conditions, particularly after eye surgery. It is interesting to note the long clearance time of this drug in dogs compared with that in man (under six hours), given by Micks (1944).

The intravenous therapeutic ratio of " Kemithal " to " Pentothal " is given as 7.2 : 4, which accords with our findings, so that " Kemithal " is about half as active, weight for weight, as " Pentothal " (Carrington and Raventos, 1946). We think this reduced activity is more than counterbalanced by its higher induction safety, especially in toxic cases; less respiratory depression; better abdominal relaxation, and the quick and quiet recovery. The respiration averages given do not show the absence of Cheyne-Stokes breathing with this drug, but the narrow range of respirations and pulse indicate its low toxicity. Carrington and Raventos (1946), working with rabbits, showed that " Kemithal " appears to have a greater safety factor than " Pentothal " at all levels of anæsthesia.

Summary

(1) " Kemithal " Sodium intravenously in a single dose is about half as potent as " Pentothal " Sodium or " Intraval " Sodium, and about the same as " Nembutal." It has a higher therapeutic ratio.

(2) The period of surgical anæsthesia given by similarly active doses was 23 minutes for " Kemithal " and " Pentothal "; 16 minutes for " Intraval," and two hours for " Nembutal."

(3) " Kemithal " is less depressant to the respiratory centre than " Pentothal," " Intraval " or " Nembutal."

(4) Recovery is quieter and quicker from " Kemithal " anæsthesia than from " Pentothal," " Intraval " or " Nembutal." Times for complete recovery from reappearance of the pedal reflex to standing were: " Kemithal," 37 minutes; " Pentothal," 4½ hours; " Intraval," 3¾ hours; and " Nembutal," 16¼ hours.

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TABLE OF ANÆSTHESIA DATA (AVERAGES)

Anæsthetic	Quantity to produce surgical anæsthesia (grains)	Signs of anæsthesia from induction (min. sec.)					Respirations: pulse (range) after induction (min.)					Recovery periods (hour. min.)				After effects
		Yawning and licking	Relaxation tongue and jaws	Loss of corneal reflex	Loss of pedal reflex	Induction time (min./sec.)	Immediately	15	30	60	120	Duration of surgical anæsthesia (hour. min.)	Pedal reflex	Corneal reflex	Standing	
"Kemithal" Sodium	6	2.00	2.00	2.00	2.00	2.43	15 : 96 (11/20) : 96/102)	17 : 110 (14/20 : 98/128)	19 : 121 (14/24 : 112/128)			.23	.23	.24	1.00	Nil
"Pentothal" Sodium	3	1.11	1.24	1.31	1.18	2.00	22 : 107 (9/60 : 70/180)	22 : 101 (12/60 : 80/120)	21 : 102 (12/52 : 80/120)			.23	.23	.24	5.00	"Running" and depression
"Intraval" Sodium	5	1.23	1.41	2.11	2.15	2.00	17 : 106 (12/22 : 84/120)	17 : 104 (12/22 : 84/122)	17 : 104 (12/22 : 88/122)			.16	.16	.17	4.00	"Running," howling and depression
"Nembutal" Sodium	6	1.40	2.00	3.00	4.00	4.00	16 : 114 6/30 : 86/160)	14 : 109 (6/24 : 84/142)	13 : 110 (6/28 : 32/160)	13 : 112 (4/28 : 40/140)	13 : 114 (4/26 : 84/140)	2.00	2.40	2.40	19.00	"Running," howling and depression

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CLINICAL ARTICLES

PEROSIS IN DUCKLINGS COMPLICATED BY A RIBOFLAVIN DEFICIENCY

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THE rearing of poultry on an intensive system in many tropical regions presents numerous nutritional problems. The protein and vitamin requirements of growing poultry are high and the mineral content of the ration must fall within certain limits. It is most difficult and often impossible to meet these needs with locally grown foodstuffs which tend to be high in carbohydrates and have a low protein and vitamin content. If an attempt is made to rear the birds on an inadequate diet the consequences may be disastrous.

The 150 ducklings were imported by air, "as day-olds" and arrived in very good condition, only three being found dead on arrival. There were three breeds, Khaki Campbells, Aylesbury and White Pekin. They were housed indoors in batches of approximately 50 under good conditions on concrete floors with chaff litter and were fed on the ration shown in Table 1, given as a dry mash. There was no access to direct sunlight, soil or grass. Each pen was supplied with a hover, heated by a kerosene lamp. The heating source was necessary to counter the sudden drop in temperature which is liable to occur during rain showers and at night. For most of the day the room temperature was maintained in the region of 90° Fahrenheit, which is the accepted temperature requirement during the first week of life. After two weeks the supply of dried milk and fish meal ran out and no replacement was found. The maize and soya beans were increased to make up the ration. During the first three weeks the ducklings thrived well with only an occasional death from misadventure.

The birds were examined at a month old just before the great majority of the deaths took place. By referring to Table 2 it can be seen that the Khaki

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Campbells had already begun to die. At this time changes on the legs could be observed from outside the pens. About one-third of the ducklings were affected. By individual examinations various stages of the condition could be seen. In the earlier stages enlargement of one or both hock joints was apparent and this was followed by subcutaneous hæmorrhage over the joint. The metatarsus then became twisted out of alignment with the tibia and the articular cartilages and gastrocnemius tendon were displaced. In a few birds there was complete dis-articulation of the joint. Several birds showed paralysis of the digits but spastic curling of the toes was not observed. Ducklings which died showed no premonitory clinical signs of note. They were found very weak and succumbed in a matter of hours or were found dead.

Post-mortem examinations were carried out on dead birds from all three breeds. Fatty degeneration of the liver was a constant feature and in a few cases petechiæ were found in the heart near the coronary sulci. Bacteriological examination of swabs taken from heart blood, liver and spleen failed to show the presence of micro-organisms. Microscopical examination of fæces did not reveal the existence of coccidial oocysts. The leg changes already described were encountered.

A batch of chicks which had been reared on the same ration as the ducklings had shown " curled-toe paralysis " and a number of deaths had occurred. This pointed to the perosis being complicated by a Riboflavin deficiency.

The Riboflavin, Choline and Manganese content of the ration was calculated from tables giving values for the individual constituents. Some variation in the figures given by different reference books was found but those were not large enough to influence conclusions which could be shown from them. It was not found possible to obtain values for all the constituents in respect of Choline and Manganese. These figures are given in Table 1.

The ration is deficient in Riboflavin and Manganese and it is most probable that it is also low in Choline.

A deficiency of Riboflavin or Choline or both could be responsible for fatty changes in the liver. The primary cause of death was likely to be the the deficiency in Riboflavin.

On the occasion that the ducklings were first examined it was advised that they should be moved on to grass immediately to provide a good source of Riboflavin and Choline. This took place the following day but was too late to prevent the severe mortality which ensued. Most of the surviving birds show leg changes to some extent.

To prevent the disease occurring in future batches of young poultry it has been suggested that 5 per cent of dried yeast is added to the ration and Manganese Sulphate be added at the rate of six ounces per ton of ration. The birds should also be put on to grass at three weeks of age.

The cost of the yeast will be fairly high but as the values for the factors in question, particularly Riboflavin, fall so far short of the accepted require-

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(*Article by Turton, page 171*)

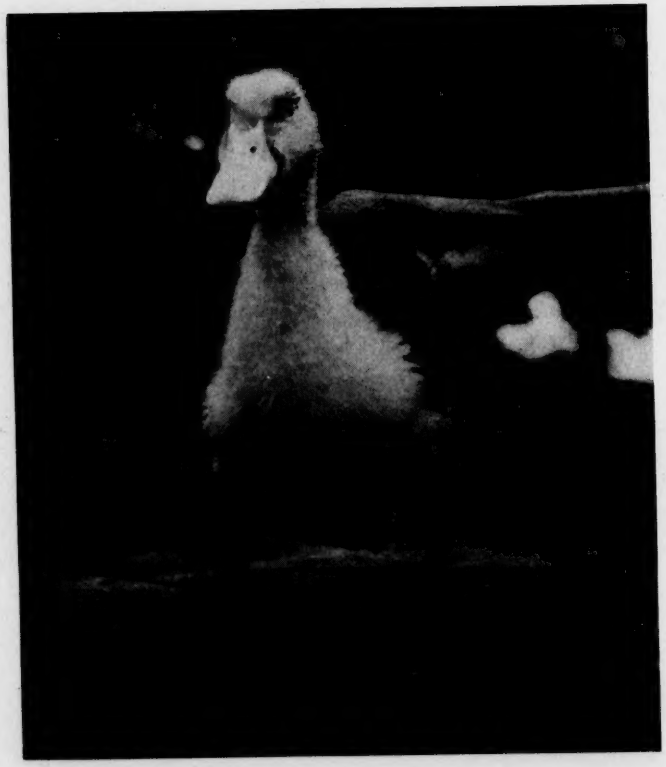

FIG. 1
Early stage.—Swelling of the hock joint with twisting of the metatarsus producing a bow-legged appearance.

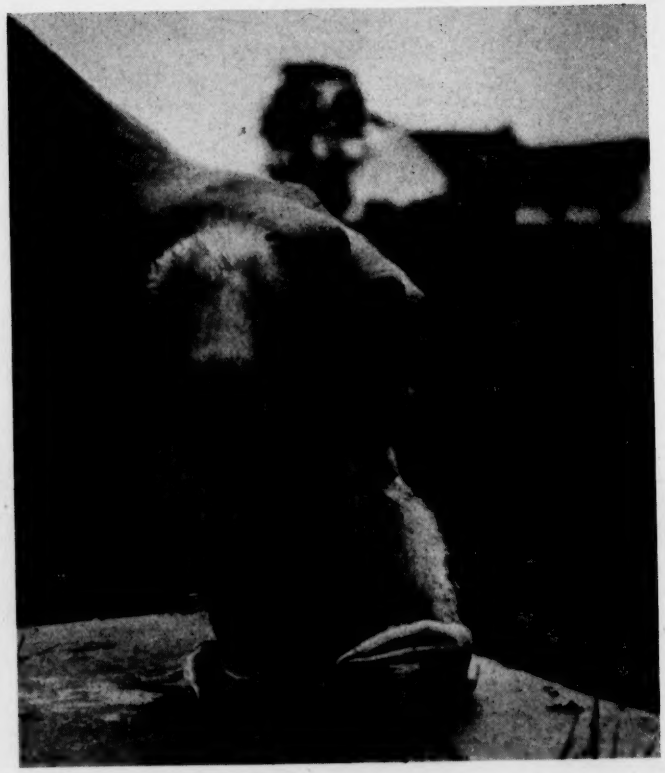

FIG. 2
Late stage.—Dislocation of the tibio-metatarsal articulation.

FIG. 3
Complete dislocation of the tibio-metatarsal articulation.
(*Article by Turton, page* 171)

FIG. 1
Myiasis (*Chrysomyia bezziana*) in a dog.
(*Article by McGaughey, page* 175)

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ments, it would be most difficult to bring this ration up to standard without it.

As other foodstuffs are made available an attempt will have to be made to compound a more satisfactory ration.

Summary

(1) A condition of young ducklings is described associated with leg changes and high mortality at four to five weeks old.

(2) The amounts of Riboflavin, Choline and Manganese in the ration are discussed.

(3) Preventive measures are suggested.

Acknowledgment

The author wishes to thank Mr. S. Simpson, M.R.C.V.S., Director of Veterinary Services, Gold Coast, for permission to publish this article.

TABLE 1

	%	Riboflavin in mg./lb. of foodstuff	Riboflavin in mg./lb. of total ration	Choline in mg./lb. of foodstuff	Choline in mg./lb. of total ration	Manganese in mg./lb. of foodstuff	Manganese in mg./lb. of total ration
Kokonte (Sun Dried Cassava)	10	Nil	Nil	No figs. available		No figs. available	
Palm Kernel Meal	15	Nil	Nil	No figs. available		90	13.5
Maize (mixed)	35	1.9	.665	100	35	2.5	.875
Groundnut Cake	10	.2	.02	No figs. available		15.0	1.5
Soya Beans	10	1.2	.12	1300	130	13.4	1.34
Fish Meal (poor quality) ...	10	.1	.01	1000	100	10.0	1.0
Dried Milk	5	1.5	.075	500	25	.18	.009
Minerals	5	Nil	Nil	Nil	Nil	Nil	Nil
Total	100	—	.89	—	290	—	18.22

Requirements of growing poultry: Riboflavin, 1.6 mg./lb. of food; Choline, 700 mg./lb. of food; Manganese, 25.2 mg./lb. of food.

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TABLE 2

Mortality Rate.

Days	Date	Khaki Campbell	Aylesbury	White Pekin	Total
1 ...	July 9	1	—	—	1
2 ...	10	2	—	1	3
3 ...	11	—	—	—	—
4 ...	12	—	—	—	—
5 ...	13	—	—	—	—
6 ...	14	—	—	—	—
7 ...	15	—	—	—	—
8 ...	16	—	—	—	—
9 ...	17	—	—	—	—
10 ...	18	—	—	—	—
11 ...	19	1	—	—	1
12 ...	20	—	—	—	—
13 ...	21	—	—	—	—
14 ...	22	—	—	—	—
15 ...	23	—	—	—	—
16 ...	24	—	—	—	—
17 ...	25	—	—	—	—
18 ...	26	—	—	—	—
19 ...	27	—	—	—	—
20 ...	28	1	—	—	1
21 ...	29	—	—	—	—
22 ...	30	—	—	1	1
23 ...	31	1	1	—	2
24 ...	August 1	—	—	—	—
25 ...	2	2	—	—	2
26 ...	3	2	—	—	2
27 ...	4	1	—	1	2
28 ...	5	1	—	—	1
29 ...	6	3	2	—	5
30 ...	7	12	1	—	13
31 ...	8	5	2	—	7
32 ...	9	5	2	3	10
33 ...	10	6	5	3	14
34 ...	11	3	2	4	9
35 ...	12	1	6	5	12
36 ...	13	1	5	4	10
37 ...	14	1	1	2	4
38 ...	15	—	1	2	3
39 ...	16	—	—	1	1

MYIASIS (CHRYSOMYIA BEZZIANA) IN A DOG IN CEYLON

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Subject : Alsatian Male, four years, guard dog in R.A.F. camp on the coast of Ceylon.

History : Bred in England, taken to Singapore in 1952; after two months stay in Malaya brought to Ceylon. Had lost condition in Malaya and developed dermatitis. A patch of skin over the sacrum had been shaved by the attendant who later noticed a conical swelling developing; this swelling enlarged to about three inches in diameter. The apex of the swelling ruptured and several maggots were detected. They were removed and placed in preservative.

Clinical Examination : The swelling with a small crater-like opening (Fig. 1) was found to contain five more larvæ. The temperature was normal, there were no microfilaria nor protozoal parasites in the blood. The fæces contained numerous hookworm eggs and also coconut fibre.

Treatment : The " abscess " had been treated previously with antiseptic lotion. " Lorexane " cream (I.C.I.) was smeared inside the cavity, anthelmintic treatment and laxative pills containing Fe, Cu, Mn and Ca were administered.

Remarks : The larvæ were identified as those of *Chrysomyia bezziana.* This fly is known as the Screw-worm Fly of the Old World. According to Patton and Evans (1929) the adult is never seen in nature and specimens have to be obtained by hatching out pupæ. Unfortunately, in the present case, the larvæ had been killed by the antiseptic lotion. According to Patton and Evans (1929) " the female flies oviposit in or near living tissues, attracted either by the smell emanating from shed blood or by that from diseased tissues. Their larvæ do not penetrate unbroken skin. They cause all kinds of cutaneous and wound myiasis as well as nasal, aural, ocular and vaginal myiasis." The infection is always a multiple one (cf. with *Cordylobia anthropophaga,* the " Thumbu Fly " of tropical Africa).

Patton and Evans state that whereas myiasis due to *Chrysomyia bezziana* is frequently found in animals and human beings in India, it is found only in animals in Africa and the Philippines. Bayer and Talwatte (1952) described two cases of myiasis (*Chrysomyia bezziana*) in human beings in Ceylon and have devised a method of attracting and trapping the larvæ when they are in inaccessible cavities; the method is to insert plugs of cotton wool soaked in dextrose solution. The larvæ are attracted and adhere to the plugs.

Summary: A case of myiasis (*Chrysomyia bezziana*) in a dog has been described.

Acknowledgments : Thanks are due to Dr. S. H. Jayawickreme, Division of Entomology, Medical Research Institute, Colombo, who identified the larvæ.

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REVIEW

BANHAM'S THE VETERINARY SURGEON'S VADE MECUM, 8th edition, by W. A. Greig. Bailliere, Tindall & Cox. Price 17s. 6d. net.

DESPITE an external metamorphosis from a "Veterinary Posology" to a "Veterinary Surgeon's *Vade Mecum*," the essential features of the 7th edition of Banham are retained in the text of the new edition.

Admirers of the earlier editions, the first of which appeared as long ago as 1887, will find that classification and regrouping has been extensive though rational, with much pruning of, dare it be said, obsolete detail. As a practitioner's guide there is patently much more pruning that could be done, particularly with regard to such tables as those of Incubation Periods and Common Animal Parasites, but on the whole the editing has been very satisfactory.

Old users of the book will also find that much new material, such as the chapters on milk and urine tests, and the various allergy tests, has been added which effectively stamps the book as modern. The inclusion of dosage tables for such accepted and widely used drugs as the sulphonamides, penicillin, the barbiturates and others, is timely and useful, although the Pharmaceutical Society may view with disfavour the expression of sulphonamide doses in grains per lb. body weight! However, practitioners will, without doubt, appreciate the compiler's decision in this respect.

Faults are very few and of little significance, several appearing to be in the nature of printer's errors only.

In short, Alastair Greig, has achieved his aim in modernising Banham for the practising Veterinary Surgeon without deleting any useful information, and it is certain that the book will continue to enjoy its perennial popularity.

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